ENVIROTOXICOLOGY

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Health Effects on Nearby Residents from the Koppers Grenada Plant Operations

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Introduction:

The current Koppers Grenada, Mississippi Tie Plant was constructed in 1904 to treat railroad cross ties for the Illinois Central Railroad (Figure 1). The plant has been in continuous operation since that time. While the plant was originally located approximately four miles south of the town of Grenada, Mississippi; over the years a small community, including a school, was constructed next to the 171-acre plant. The plant employs approximately 60 people locally, and produces utility poles, piling, and railroad ties for various customers in Mississippi, the eastern USA, and for export. At least until the mid-1990's approximately 80 percent of the wood treated at the plant was purchased from within Mississippi. Until recently, these wood products were pressure treated with creosote or pentachlorophenol wood preservative solutions.[1]

At the Grenada Koppers plant, wood preservative chemicals, creosote and Pentachlorophenol (PCP) are impregnated into wood stock under high temperature and pressure. Following treatment, the wood stock is stacked and allowed to air dry. This plant has extensive acreage devoted to drying the wood stock. Excess chemicals from the wood treatment processing are discharged from the processing plant into the ambient atmosphere, allowed to evaporate into the air and drain off of the stored wood stacks into the soil (Figure 2). Waste chemical fluids from the treatment plant are directed to waste discharge ponds where evaporation is allowed to take place (Figure 3). Multiple small ditches guide excess chemicals from the wood stacks into small creeks, which eventually are discharged into a river. Residents from the town of Grenada use this river to supply

their potable water. The town's resident's exposure pathways include air, soil, surface water and possibly drinking water.

Figure 1. Image of the Koppers Plant, Grenada MS.

Figure 2. Chemicals leaching into local streams and creeks near Koppers Plant.

Figure 3. Chemicals leaching into local streams and creeks near Koppers Plant.

The chemicals used as wood preservatives at this plant have many well-known harmful health effects upon exposure to both animals and humans, including cancer. Creosote is a complex mixture that contains numerous polycyclic aromatic hydrocarbons (PAHs)[2] and pentachlorophenol is contaminated with polychlorinated dioxin and furans.[3] The residents of Grenada have been exposed to significant amounts of these chemicals from the Koppers Grenada Plant. Their exposure to these chemicals (dioxin, PCP, and adduct testing) is higher than the general public (NHANES) and comparison populations. The testing of the environment by 3TM shows significant quantities of polycyclic aromatic hydrocarbons including Benzo(a)pyrene, Benzo(a)anthracene, Indeno (1,2,3-cd)pyrene, and dioxins including 2,3,7,8-TCDD in the dust and soil in the nearby residents' homes and at the nearby local elementary school.

Exposure:

The plant has caused severe pollution in the surrounding neighborhood. The pollution arise from the normal operations and from accidental releases. One of the most significant sources of exposure was the combustion of wood waste in their boilers. A significant percentage of the waste wood had been treated with PCP and creosote. Such wood burning will create a large amount of dioxins to be released into the air. Steam is produced to provide process heat for wood preserving processes and for kilns to dry wood prior to treatment. Additionally, a turbine and electric generator utilize excess steam to provide electricity for the Plant. Beginning in 1904 ash waste from the boiler and wood waste was spread on the property in several different waste sites: (1) Boiler Ash Landfill; (2) South Waste Piles; (3) North Waste Piles; (4) Surface Impoundment Area; (5) Wood Disposal Area, and (6) Spray Irrigation Field. The history of the plant with regard to the exact locations of discarded ash waste sites is not entirely clear by the records reviewed to date. Liquid wastes from drip pads were allowed to run into ditches, which carried the liquid waste off site.[4]

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A Mellons wood fired boiler was installed in 1978 to provide process heat and utilize wood waste fuel instead of fuel oil or natural gas. A small turbine and generator were installed to reduce electricity cost by utilizing excess steam.

Koppers boiler was permitted to burn wood preserving process waste, containing pentachlorophenol and/or creosote, as fuel additive to the wood waste fuel about 1985. Burning of process waste as fuel additive continued until such wastes became listed

hazardous wastes in June 1991. At that time, Koppers stopped such activity until a RCRA hazardous waste permit could be obtained. Koppers attempted to obtain a RCRA permit under the Boiler and Industrial Furnace (BIF) regulations beginning in late 1991. Due to the lack of permitting progress and a negative regulatory climate for any hazardous waste combustion systems, the decision was made in late 1993 to withdraw Koppers' BIF application.

As of March 1994, all such hazardous process wastes, which are generated by Koppers, are now, and will continue to be, disposed off-site. As of March 1994 Koppers is no longer seeking any permit to burn hazardous wood preserving process wastes.[1]

Disposal of the treated wood products at the end of their useful lives has become a costly issue for Koppers' customers. Although such materials are clearly not listed (as of March 1994) or characteristic hazardous waste, disposal options have become limited and costs are rapidly increasing, especially with landfill space becoming more limited. For the last several years, Koppers has responded to their customer needs with a program of receiving such used treated wood and recycling it for energy recovery in its Grenada industrial boilers. Currently (March 1994) Koppers operates three boilers, which are permitted to burn treated wood, and a fourth has obtained a permit. Koppers requested approval in March 1994 to utilize waste treated wood containing creosote and pentachlorophenol preservatives, as a primary boiler fuel. (There are possibly a total of five boilers according to other sources.)

Koppers request for permitting a fourth boiler states in part: "Koppers is now paying a significant price for wood fuel for our boiler. We believe what use of creosote treated wood fuel will significantly lower that cost. Most railroads and utilities now dispose of used wood by landfilling to a substantial cost. The reduced disposal cost and elimination of long term liability resulting from burning the used wood as fuel at Koppers' boiler would be a substantial benefit to those customers." Koppers stated also it believed that creosote and pentachlorophenol treated wood waste can be burned in a boiler without causing any adverse human health or environmental impacts or any significant increase in emissions.[1]

1909-1972

Creosote Gallonage Sales to Koppers' Grenada Plant from 1909-1972 = 9,566,069 M gallons.[5]

1980's

This file contains a significant amount of information in memo form indicating that between the years 1981-88 Koppers was not successful in obtaining permits to discharge wastewater in concert with current permitting regulations.[6] At the same time, Koppers was applying for modifications to be made to their main boiler, in order to be able to use "treated" waste wood products for fuel to enable Koppers to increase their throughput at the plant. In a memo, dated 7/31/86, which was a report of a meeting held between Koppers, et.al. and the MDNR personnel, several key points were made: (1) Koppers needed to work with both the City and State to obtain a POTW permit, (2) Koppers

acknowledged the Grenada plant did not indicate previously they wanted to be included in the POTW permitting system and (3) in the proposed application (by Koppers) it was assumed that the present Grenada POTW plant had excess capacity and could possibly handle the Koppers' Grenada plant wastewater without further treatment. After reviewing the State's and City's comments and concerns, it became apparent that this was not the case. The memo contains two additional typewritten pages of concerns by both the City and State regarding these matters.[7] On page 4 of the Application for a State Operating Permit[8] Koppers discloses that in addition to standard toxic pollutants that will be contained in the discharge water regulated under the CWA of 1977, the following chemicals will be included in the discharge water: Octochloro-dibenzo-p-dioxin, heptachloro-dibenzo-p-dioxin, octochloro-dibenzo-p-furan, and phenanthrene. The attachments to this application[9] provide wastewater analysis reports dated 8/19/85. The memos and reports in this 472-page section are not in any order and must be viewed on an individual basis.

August 6, 1981

Interoffice memo to key Grenada staff which states in part, "Reports from Guardian Systems and Air Quality Engineering Section at Monroeville Research Center represent all the data we have on burning creosote and penta sludge in Wellon's type boiler systems. They indicate if sludges are burned under the conditions shown, no harmful effects will be created by incineration." [10]

February 1982

Letter from D.N. McLeod, MSDNR, dated 2/22/82 regarding Koppers' request to begin blending wood preserving waste stream with wood chips to fuel their boiler. One complaint had already been received from the community. Mr. McLeod requested meeting with Koppers' staff to determine how to "handle this matter." A change in Koppers' Operating Permit would be required. A copy of an internal Koppers memo dated 2/26/82 from W.J. Baldwin to T.A. Marr, states in part: "I have attached the Mississippi Bureau of Pollution Control's response (the letter of 2/22/82) to our October 16, 1991 request to have sludges in Grenada's wood-fired boiler. As I have repeatedly mentioned, the Bureau is having a difficult time resolving this issue due in part to similar requests by other treaters. In fact, Mr. McLeod states, "We have questions that are unanswered and we are concerned about public reaction to such a project in the Tie Plant area." "I am concerned that the Bureau has already received a complaint, and would appreciate any information we have in this regard." [11]

August 2, 1985

This document is a draft EIR report dated 8/2/85 by Law Environmental Services.[12] The report states on page 15, "The surface impoundment is the only unit under consideration in this Exposure Information Report." This report is part of an application by Koppers to the State to permit enlargement of their boiler operations and to increase waste discharge during plant operations. While multiple deficiencies are listed on page 2 of the report, they are not included in the final conclusion of the report, page 21, which states in part,

"The ground-water pathway is the principal avenue for potential exposure to humans to K001 waste constituents released from the surface impoundment. The county drinking water wells are located within three miles of the surface impoundment. There are no data suggesting that the public drinking water is contaminated, and the data from an existing monitoring well system do not indicate a plume of contamination exist. There is very little chance for human exposure to releases to the soil from the surface impoundment."

October 8, 1985

This set of reports entitled "RCRA Part B Application for the Koppers Company, Inc. Hazardous Waste Management Facility Grenada, Mississippi Surface Impoundment" [13] contains several items of interest: (1) memo dated 6/14/85 from EPA to Assistant Regional Counsel – Region V (Grenada plant's EPA region) stating "kick-back" drippage areas (the ground area around tracts or other areas outside of the immediate treating cylinder area where drippage of the immediate treating cylinder area collects from newly treated wood) is a solid waste management unit, subject to the corrective action requirements of the 1984 RCRA amendments (hazardous waste). Additional language also includes in this change in definition, Grenada's sprayfield for liquid wastes, (2) Grenada's management strongly opposed these EPA interpretations (which were accepted by the Mississippi Dept. of Natural Resources, and via two sources (a) Keystone Environmental Resources (3/09/88), and (b) law offices of Weil, Gotshal & Manges (02/26/88), wrote strong opinions to MDNR disagreeing with this decision and declared the "Agency's (EPA) conduct illegal."

March 1987

This is a report of both ground and surface water conditions at Koppers' Grenada Plant.[14] The report covers all acreage of the plant, its present operating wells, and waste discharge locations. The report contains detailed maps of all aspects of the plant. The report presents detailed descriptions of contaminated and potentially contaminated sites on the property. The section on Recommendations strongly states monitoring wells should be placed at strategic locations on the property, many of which are close to the plant's border with the community's housing and school properties. Review of the most recent data available reveals some monitoring wells were put in place, but it appears the most important wells, which could monitor potential contamination of community aquifers, were not placed. Strong recommendations were made for re-sampling of existing well sites, the type of monitoring to be made and a time line in which to carry out these recommendations. However, the records reviewed to date do not indicate if these recommendations were carried out. Of critical importance is the conclusion the plant survey found multiple contaminated sites, which directly or indirectly were identified to be polluting groundwater.

August 17, 1987

In a letter[15] dated August 17, 1987, Koppers requested the MS State Attorney to de-list Koppers' Grenada plant's boiler ashes as "hazardous waste." MS had recently declared the ash to be classified as Hazardous Waste, and had issued an immediate restraining order for Koppers to cease from distributing the ash on Koppers' Grenada's properties. If this petition was to be denied, the letter stated this issue would be appealed to the EPA for

final disposition. Note: On June 6, 1991, RCRA changed the definition of Hazardous Wastes to include Grenada plant's boiler ashes as "hazardous waste."

December 10, 1987

Internal memo dated 12/10/87 reflecting the need to repair completely the wood fired boiler. Immediate repair is indicated.[16]

May 2, 1988

Results for a boiler stack test performed during the week of 5/2/88 show that the boiler is well within compliance with regard to particulate emissions, and that the boiler effectively destroys constituents associated with using wood treating wastes as fuel additive material." [17]

August 30, 1988

Letter[18] from RCRA Branch of EPA to Chief of Hazardous Waste Branch, Mississippi Dept. of Natural Resources (DNR) regarding penalty calculations for Koppers Company, Inc., located in Tie Plant, MS. Significant statements include the following: "These penalties were calculated to give an idea of the penalty that EPA would assess and what we would like to see assessed by MSDNR." "The facility has been operating a sprayfield which is subject to RCRA regulation. The facility has been notified several times throughout the past seven years that the unit was regulated and not included on the original Part-A Permit Application. A Part-B Permit Application has also been called for several times. Koppers has a history of not complying with the regulations, and delaying

on submittals required by EPA and MSDNR. Based on these facts EPA Suggest MSDNR assess a penalty to Koppers for operating the sprayfield without having interim status or a permit." "There are two penalties calculated. One is the amount of penalty for the use of the sprayfield since November 19, 1980, which was the date set for Part-A's to be filed. Since Mississippi feels that the decision to regulate sprayfields was unclear at that time, a second penalty was calculated. The date used for the first day of non-compliance in the second penalty is April 26, 1984, which is the date Mississippi first informed Koppers that the sprayfield was regulated. An inspection conducted by John Herman on April 26, 1984, of MSDNR, documents that the sprayfield is a land treatment unit and that was not included in the Part-A submitted by Koppers. The two amounts of penalty calculated were $713,750 and $400,250 respectively. EPA feels that the violation committed by Koppers warrants a substantial amount of penalty and would like to see MSDNR assess such a penalty."

June 23, 1988

Internal memo to Grenada staff stating Grenada Plant was going to withdraw from using pentachlorophenol in its operations. Neither date of withdrawal, nor any follow-up information was included in this memo.[19]

September 1988

This is a report covering the wood boiler performance for September 1988. Inspection of the boiler proved the boiler to be in a severe state of disrepair, requiring immediate remediation for multiple problems.[20]

1989

Koppers Fuel Additive Program is detailed in these 644 pages of information. This section contains records of Grenada plant drum inventories of fuel additives received from various Koppers' plants. The majority of the records are from 1988-89; however, there are multiple receipts for transportation and other activities that are dated from 1983-1989. Scattered throughout these 644 pages are memos and other written documents having to do with the fuel additives program, including problem solving on the equipment used, permitting documents, and certification documents of various kinds. A memo dated 4/27/89 (050068) states in part: "Wastewater will start to be treated Friday, April 28, 1989....I do not want any receiving parties to discount the future addition of an above ground metal separator and a fiber glass pump tank going to the City and one from the separator to the biological end (P.H. adjustment tank). This will not hinder a start-up but must be considered for the future." There are multiple documents dealing with significant plant alterations that had to be made to keep the plant in permit compliance, as well as to expand their operations. Many of these memos are brief, but describe significant issues that the plant had to deal with in undergoing the construction of a new boiler operation to burn waste fuels to provide pressure and steam for their existing and expanded operations at Grenada. A memo dated 9/21/89 from Beazer to the RCRA TSD Branch Hazardous Waste Division MDNR (049894) states in part: "The final cap components for closure of the surface impoundment are currently being placed. We have not as yet received the Appendix IX results from the groundwater sampling round completed in June, 1989 and will submit those to you when available." In my opinion, the results of the groundwater sampling round should be obtained and reviewed if at all

possible. It is my impression once all caps are in place; Beazer has no further duties on the plant property. The results of a safety audit, in a memo, dated 9/18/89 (049901-049907) provides a detailed list of corrections recommended for the plant on that date.[21]

Koppers - Internal Memo – December 13, 1991

This memo[22], written to staff, confirms Koppers has been continuously operating the boiler on its' Grenada property for many years. It states in part, "Since 1982 Koppers has used wood preserving process wastes from other Koppers owned facilities as a fuel additive to this boiler. The fuel additive program at the Grenada boiler has been valuable to Koppers by providing an alternative to land disposal of our process wastes while reducing the Grenada plant's need to purchase supplementary fuel. These process wastes were not RCRA hazardous wastes." However, the memo goes on to state due to changes in new Boiler and Industrial Furnace (BIF) regulations, 40 CFR 266, permitting of the new boiler will be necessary. The ash from the boiler was changed on June 6, 1991 by new RCRA regulations to a category of hazardous waste.

August 1992

This is a very important permit application by Woodward-Clyde Consultants on behalf of Koppers. This Class 3 Permit Modification for Boiler and Container Storage, dated August 1992. Important sections of this permit applications are quoted as follows: "KII (on Grenada property) is a generator of hazardous waste resulting from the wood preserving and coal tar processing operations. KII is also buying boiler fuel for the Grenada plant boiler for process heating requirements. The ability to utilize KII generated

hazardous waste, as fuel will significantly reduce treatment and disposal costs while also utilizing the fuel value of those wastes. Since the boiler and storage facility are not currently included in the RCRA permit, the permit needs to be modified to allow these operations. KII will receive and store hazardous wastes from other KII generating plants (Koppers facilities), which can be burned for fuel value in the Grenada plant boiler. Hazardous waste storage will be conducted in the container storage facility, prior to the wastes being transferred to the boiler for burning. Authorization to burn KII generated wastes will allow resumption of a waste management technique which KII (at the Grenada plant) has been utilizing since 1982 and which was disrupted by the EPA's listing of wood preserving process wastes as hazardous waste in June 1991. KII has since been paying disposal firms to treat and/or and dispose these wastes. Koppers is the operator of two hazardous waste units on the facility, the hazardous waste storage unit (S1) and an industrial boiler, utilizing hazardous waste as fuel (T04). Koppers is the current owner and operator of the wood preserving business on this site. The wastes to be accepted from the other wood preserving plants are generated by the same processes as are currently operating at the Grenada wood preserving plant. The wastes to be accepted from Koppers tar plant are from a specific source, the production of phthalic anhydride."
23

August 19, 1993

This section deals with the decision-making process between Koppers, Inc., Beazer, PLC and the EPA regarding allowing the Grenada permission to act as a hazardous waste disposal site, and whether Grenada wished to do so. On June 6, 1991, new RCRA

hazardous waste listings became effective which defined certain wood preserving wastes utilizing pentachlorophenol as FO32 hazardous wastes. Other wood treating wastes from processes utilizing creosote and CCA (chromated copper arsenate) processes were also listed, but did not become effective until adoption by the authorized state. On August 21, 1991, regulations for the burning of hazardous waste in boilers and industrial furnaces ("the BIF rule") became effective. Thus, in a period of less than three months, KII's plans to burn hazardous waste in its Grenada boiler were impacted by the FO32 listing and then the BIF rule. On 12/16/91, the MDEQ received a request from Koppers to evaluate a proposed operational plan that Koppers' Industries hoped to be able to implement at its facility in Grenada, MS. The plan called for pentachlorophenol and /or creosote process wastes, generated at other Koppers' facilities located throughout the U.S., to be burned in the Grenada facility's industrial boiler. The state would require the facility to reclassify the plant as a commercial hazardous waste facility, and as such would subject the facility to all rules and regulations that govern this type of operation. An untitled and undated typed statement as the first page of this section explains Koppers' problem and I quote: "The BIF hazardous waste permit offers significant potential benefits and liabilities. We need to carefully reevaluate the potential benefits, especially the continuing avoided cost of phthalic waste disposal. The greatest liability I see is the possibility of fines for non-compliance. Even with great vigilance, an unfriendly agency could potentially uncover noncompliance, which could, in the current anti-inceration environment, lead to large fines." In a memo dated 6/9/92 to the RCRA and Federal Facilities Branch, Koppers states they have decided to proceed with obtaining a RCRA permit to operate the industrial boiler at the Grenada plant as a hazardous waste facility. In a memo dated

4/23/92, the second paragraph describes ownership of hazardous waste impoundments at the Grenada plant and is summarized as follows: "Koppers Company, Inc. was a large, diversified corporation when it was acquired by Beazer PLC in 1988. Beazer sold the coke, tar refining and wood treating businesses to a management group in a highly leverage buyout. Beazer retained the environmental liabilities, as they existed at the time of the buyout. With particular regard to the Grenada plant, Beazer retained exclusive responsibility for the surface impoundment located there. KII never operated the surface impoundment and Beazer has closed that unit. Beazer's exclusive interest is to close each unit so as ultimately to terminate its responsibilities at each location.[24]

1986-1994

A memo dated 3/07/94, requesting of the MDEQ a modified operating air permit provides a concise history regarding plant operations as pertains to the air permitting process. Summarizing: The Grenada Tie Plant was built in 1904 to treat railroad cross ties for the Illinois Central Railroad. The Wellons wood fired boiler was installed in 1978 to provide process heat and utilize wood waste fuel instead of fuel oil or natural gas. A small turbine and generator were installed to reduce electricity cost by utilizing excess steam. Koppers was seeking to modify their permit to (1) eliminate the provision allowing burning of wood preserving process waste as fuel additive and (2) allow use as a primary fuel used treated wood containing creosote or pentachlorophenol. In addition, the existing permit, which, by letter dated September 26, 1990, was further extended indefinitely pending agency action, should be renewed to reflect our current operation. Koppers boiler was permitted to burn wood preserving process waste, containing

pentachlorophenol and/or creosote, as fuel additive to the wood waste fuel about 1985. Burning process wastes as fuel additive continued until such wastes became listed hazardous wastes in June 1991. At that time, Koppers stopped such activity until a RCRA hazardous waste permit could be obtained. Koppers attempted to obtain a RCRA permit under the Boiler and Industrial Furnace (BIF) regulations beginning in late 1991. Due to lack of permitting progress and a negative regulatory climate for any hazardous waste combustion systems, the decision was made recently to withdraw our BIF application. All such hazardous process wastes, which are generated by Koppers, are now, and will continue to be, disposed off-site. Koppers is no longer seeking any permit to burn hazardous wood preserving process wastes. Disposal of the treated wood products at the end of their useful lives has become a costly issue for Koppers' customers. Although such materials are clearly not listed or characteristic hazardous waste, disposal options have become limited and costs are rapidly increasing. Koppers has been responding to our customers' needs with a program of receiving such used treated wood and recycling it for energy recovery in our industrial boilers. Koppers now operates three boilers that are permitted to burn treated wood and a fourth has obtained a permit, although facility modifications have not yet begun. With this application, Koppers is requesting approval to utilize waste treated wood containing creosote and pentachlorophenol preservatives, as a primary boiler fuel. We expect that you (MDEQ) will have concerns about potential emissions resulting from burning of treated wood. Koppers shares this concern, but believes that creosote and pentachlorophenol treated wood waste can be burned in a boiler without causing any adverse human health or environmental impacts or any significant increase in emissions. Note: All communications address the question of air

emissions. There are no memos, letters, etc. addressing the solid waste discharge issues (ash and/or sludge) from these boilers in this section.[25]

May 6, 1996

This Stack Testing Report from Emission Point AA-001 Wood Fired Boiler presents the results of a two-day test of boiler emissions during "low" and "high" fire test runs. In Appendix E, GCMS Analysis, no data is present. A significant amount of test data is present in the stack testing report-dated 2/20-21/96. The report contains no written interpretation of the data obtained during this "test" run of two days. A letter dated May 6, 1996, contained in this section to the MSDEQ state the test results, "as documented by the report, most parameters are well within the requirements of the permit. Issues related to some other parameters need to be addressed." The letter states Koppers "recognizes that emission levels indicated by the test for some constituents exceed levels allowed by the permit" and request "minor permit modifications to be made." Specifically revise the NOx emissions limitation; revise the emission limitation for Carbon Monoxide, revise the temperature limitation and revise the description of the boiler itself.[26]

July 18, 1997

Titled "Major Air Pollution Source Annual Emissions 1996", this report contains total emissions of the Grenada plant for 1996 by calculation only, no actual monitoring data is included. Introductory letter dated 7/18/97 indicates emission rates differ from permitted rates and requests a meeting to discuss the differences between the allowed by permit and actual emissions.[27]

1996-1998

This is a compilation of quarterly reports made to the MSDEQ regarding operations records of the boilers employed by Koppers at the Grenada Tie Plant on the dates 1996-1999. Letter 6/16/97 from MSDEQ to plant stating complete inspection revealed no apparent air pollution problems {[110107] Letter 2/9/98 from MSDEQ to plant stating complete inspection revealed no apparent air pollution problems [110108]}. Email dated 7/28/98 states "numerous instances where the temperature was below the limit." This presumably allowed illegal discharge of CO or sulfur oxides into the atmosphere. Contains daily and hourly reading of opacity measurements in stacks at sites AA-001 and AA-002 for first three quarters of the year. Memo 4/10/97 [109950] to D. Burchfield, Air Facilities Branch, MS DEQ states permit operations i.e. stack emission, were exceeded numerous times and the records are contained herein. Memo June 24, 1997 stating similar emission exceedences and rationale for the occurrences - use of " wet" untreated sawdust." Memo 7/18/97 stating exceedences have continued. Requests face to face review with MSDEQ. Report 7/28/97 demonstrating exceedences in stack discharge levels of CO and VOCs [10953] (VOCs not separated but reported as "total"). Memo 1/22/98 to MSDEQ stating "dirty lens" on opacity meter was replaced. No mention of how long the "dirty" lens went unnoticed. Memo 7/29/98 to MSDEQ re: subj. of failing temperature probes in both cells, AA-001 & 002 between dates of April 22-May 23, 1998 which resulted in lowered temperatures necessary to complete the combustion process. [109970]. Memo to MSDEQ summarizing monitoring problems during early to mid-1998. [109972] Memo August 21, 1998 to MSDEQ again stipulating trouble with boiler temperatures remaining at permitted levels. Request for face-to-face meeting. Memo

12/23/98 to MSDEQ stipulating since Sept. 2, 1998 no additional compliance issues had arisen. [110094] Memo Jan. 19, 1999 to MSDEQ stipulating problem with a leak in the CO monitor was repaired on 1/7/99. [110098] Memo to MSDEQ dated 1/29/99 stating the plant is in compliance with Operating Permit No. 0960-00012.Complete report included [110109-11026] Air emissions test - complete report dated 12/28/98. No interpretation of results is provided. [110148-110168]. Letter 1/19/99 from Koppers to MSDEQ informing them that during the stack test for the Wellons wood-fired boiler (AA-001) performed on 12/15/98, equipment problems prevented them from operating at maximum capacity. [110147].[28]

1997-2000

This section contains required Air Emissions Reporting Forms sent to the MSDEQ for the years 1997-2000. The Annual Emission Report - 2000, contains detailed information regarding how calculations were derived as reported to the State. A letter to the MSDEQ dated June 29, 1997, describes how the plant calculated sulfur emissions based upon the tons of particular wood fuel used by the boiler; untreated, Creosote treated and Pentachlorophenol treated wood (%S x tons wood = tons S). It appears no actual air measurements were ever made.[29]

June 1, 2001

This concise 29 page report summarizes the "actual emissions data 2000" from the Grenada plant in a letter to the MSDEQ dated 6/1/01. Of great importance is an accompanying document detailing how these emissions were calculated (arrived at) in

view of the fact actual monitoring of the air and waste discharges were not obtained. This document states how the wood treating industry calculates emissions from treatment plants for submission to state and federal authorities, contained in a memo dated 6/11/01.[30]

September 26, 2001

Title V - Air Permit Renewal 2001. This is a 338-page document, which contains very summarized and condensed data from emissions from 2000. The letter of introduction dated 10/28/02 states changes have been made to plant operations since the original permit application was submitted on 9/26/01. Due to these changes, additional considerations by the MSDEQ will have to be made prior to issuance of an Air Permit Renewal. The letter contains the statement "Koppers began using only untreated wood fuel in the wood fired boiler in November, 2001. This required a reconfiguration of the data submitted on 9/26/01.The replacement data is noted and included with this application. Some references are made to data dating to 1996. Some of the data in this application is entitled "potential to emit" and does not reflect actual emissions data.[31]

Summary of Exposure

The files included for review contained primarily those that dealt with actual and/or air pollution issues. Some groundwater issues were raised; however, the majority of the documents excluded the primary data sources for groundwater monitoring results. These files contained also several reports of occupational health studies and industrial hygiene studies of employees at the Grenada plant. It is apparent after reviewing the files;

Koppers Grenada Plant management faced many difficult issues in trying to meet both state and federal environmental regulations. It appears in many instances they did not operate within the parameters of their permitted operations. Great concern rests with this reviewer regarding the potential contamination of the environment, including air, water, soil and sediment that has and is occurring as a result of the continuing operation of the plant. Review of additional files and obtaining environmental measurements will assist greatly in determining the extent of environmental contamination that most likely is present in and around Koppers' Grenada Tie Plant. Air and water measurements that are in the files require interpretation by a skilled environmental engineer.

Creosote, coal tar and coal tar pitch:

A variety of different products are commonly referred to as "creosote". Creosote is a mixture of many chemicals, the exact composition depending on the source material used and a variety of other factors. Coal tar creosotes are the distillation products of coal tar starting with the high temperature treatment of coal. Coal tar pitch is a residue produced during the distillation process and a common component of coal tar creosote. The International Agency for Research on Cancer (IARC) classifies coal tar pitch volatiles as a Group 1 carcinogen (carcinogenic to humans). Coal tar creosote, coal tar and coal tar pitch are mixtures of similar compounds, all with similar properties. In general, coal tar creosote consists of at least 75% polycyclic aromatic hydrocarbons (PAHs). However, thousands of chemicals have been identified in various mixtures of creosotes. The major chemical components of coal tar creosote that are known to cause harmful health effects are polycyclic aromatic hydrocarbons (PAHs), phenol, and cresols.[32] The exact composition of the final creosote compound depends on the source material used, the distillation procedure and the intent of the maker to include or exclude various components.

There is a relative paucity of research on the health effects of coal tar creosote despite the large numbers of workers and nearby plant residents exposed to the mixtures. Even so, enough has been published to inform a thorough report by ATSDR as to the many health effects of coal tar creosote[32] and an IARC position paper that asserts: "there is *sufficient evidence* that occupational exposure to coal-tars ... is causally associated with the occurrence of cancer. There is *sufficient evidence* that coal-tar pitches are carcinogenic in

humans. Taken together, the data indicate that coal-tars and coal-tar pitches are causally associated with cancer in humans and that creosotes derived from coal-tars are probably carcinogenic in humans." [33]

In addition to the literature specifically studying coal tar creosote, research on polycyclic aromatic hydrocarbons (PAHs) is also relative.[32] For this reason, studies of coal tar creosote as well as other substances including asphalt, cigarette tar, coal tar and others that identify PAHs (especially benzo(a)pyrene) as the primary exposure should be considered to better understand the potential health effects of coal tar creosote. It must be noted, however, that the effects of PAHs is likely to vary from the reported measures when it is experienced as a component mixed with other compounds. Firstly, the presence of other compounds may have synergistic effects. Second, the level of exposure to PAHs will vary depending on the particular mix of creosote. Information on the chemical composition of studied creosotes is rarely reported.

Coal tar, coal tar pitch and coal tar pitch volatiles are used, produced or are byproducts formed in many different situations including road paving with asphalt, roofing, tobacco smoking and many other industrial and non-industrial uses. PAHs are also found in soot, air pollutants, petroleum and cutting oils.

Epidemiology of creosote associated cancer

The association between coal tar creosote and cancer is biologically plausible because of the carcinogenic components in creosote, namely benzo(a)pyrene and

benz(a)anthracene[34] but also others.[32] The associated increased rates of cancer, causes widespread concern among nearby residents of creosote waste sites.[35] This type of concern often leads to important questions that can be addressed through epidemiologic research. This questioning is often the first step to understanding serious health effects of seemingly inert or "safe" materials. In the case of coal tar creosote, the substance is far from safe. It is recognized that dermal exposure to coal tar creosote leads to skin and eye irritation, phototoxicity and cancer.[32] In addition, genotoxic effects are well documented.[32] A summary of some of the cancer associated with creosote publications are listed in Table 1.

Through an examination of epidemiologic studies, the known biological health effects from coal tar creosote and an examination of animal studies, the weight of the evidence clearly supports the existence of a causal relationship between coal tar creosote constituents and numerous adverse health effects including cancer, birth defects, premature birth, respiratory damage, skin itch, gastric upset, immune system alterations and neurological injury.

In 1980 Dusich et al published a report of an ecologic study comparing two similar communities to St. Louis Park, a community where high levels of PAHs were found in their water supply from a creosote treatment plant's waste.[36] They used a comparison group of the entire Minneapolis-St. Paul Standard Metropolitan Statistical Area (SMSA) and an matched unpolluted area. They found increased rates of breast cancer and cancers of the gastrointestinal tract in females compared the comparison groups. The authors

report this finding as particularly interesting in light of animal studies that showed increased rates of mammary carcinoma in female rats.

Several years later, a report adjusting the rate ratios by examining the prevalence of known risk factors in the exposed cases and an equal number of cases from the referent population is published.[37] The new rate estimate was less than 1.0 showing no excess risk of breast cancer in the exposed population. No examination of the measured increase risk of gastrointestinal cancer in women was attempted because one or more observations were expected to reach the level of significance due multiple comparisons and breast and colon cancer share several common risk factors.

In brief, what the authors did was apply the estimated attributable fractions from several different known and suspected risk factors (age at menarche, age at first birth, parity, age at menopause, body mass index, history of benign breast disease, family history and Jewish religion) as reported in one study.[38] The authors did not do was acknowledge and discuss the limitations and bias of this reanalysis. They mention that a necessary assumption is that the populations studied have to have relative risks similar to those described in the literature used as the source for the population's relative risks (the correction factor) and that there was an assumed independence among the relative risks. They do not discuss the possibility that the exposure initiated a mechanism of disease that increased the prevalence of the risk factors that they examined and used to adjust downward the risk ratio. For instance, it could be that exposure to low levels of PAHs in the drinking water caused women to develop benign breast disease and affected their

ability to bear children reducing the number of full-term pregnancies that they experienced. It could also be argued that women whose families (mothers, grandmothers) were also exposed to the drinking water may have had similar disease experiences due to this exposure. This analysis produces an estimated relative risk that errs conservatively toward the null value. In the interest of the public's health, this estimate should strive to err in the direction of identifying factors that contribute to or initiate poor health. To our knowledge no further action was taken to better understand the cause(s) of increased breast and gastrointestinal cancer in the women of St. Louis Park or any other women residentially exposed to coal tar creosote.

A series of studies in Sweden were undertaken to examine the possible health effects of various occupational and environmental exposures. Several disease outcomes were examined. In 1987, Flodin, Fredriksson and Persson reported the results of an examination of possible risk factors for multiple myeloma.[39] The study design used a case-referent approach. Cases were found at hospitals and were therefore both incident and prevalent cases. Controls were drawn randomly from the catchment area of the hospitals where the cases were recruited. Exposure assessment was accomplished via a mailout questionnaire. The investigation mainly focused on radiation exposure, however, other occupational exposures were established and examined including exposure to creosote. The authors report a significant association between multiple myeloma and creosote (4.7; 1.2 - 18.0) controlling for age, exposure to fresh wood, exhaust, concrete- and brickwork, sulfonylurea, gamma radiation, ex smoking, farming and gender. The authors state that creosote exposure seems to be a "potent" risk factor for multiple

myeloma and suggest that the mutagenic polycyclic aromatic hydrocarbons in creosote may explain its carcinogenicity.

The study, reported by Persson et al., was conducted similarly to the Flodin study.[40] They also found significant risk associated with creosote exposure for both Hodgkin's disease and non-Hodgkin's lymphoma. The authors note that in light of the significant association between creosote and multiple myeloma found using the same pool of controls, there exists the possibility that creosote exposure was underestimated in the control population. However, they also conclude that the existence of mutagenic constituents of creosote (PAHs) may likely cause its carcinogenic effects.

In 1992, Karlehagen et al. published a study of cancer incidence among 922 workers from 13 plants who were directly exposed to creosote.[41] The overall rate of cancer was lower compared to the national rates. This type of finding called the "healthy worker effect" is very common among occupational epidemiologic studies. It is only when individual cancer sites are examined that one is likely to find increased rates in worker populations exposed to carcinogens and even then, it is usually necessary to examine the rates using the appropriate lag time for the cancer at issue. In this case, increased rates were observed for lip cancer (SIR 2.50, P=0.05), nonmelanoma skin cancer (SIR 2.37, P = 0.02) and malignant lymphoma (SIR 1.9, P = 0.06). When 20 years of time since first exposure is considered (lag or latency), the excess risk for lip cancer, nonmelanoma skin cancer and malignant melanoma was 3.7, 3.1 and 2.2 respectively. One of the plants used

arsenic, known for causing skin cancer; however, none of the skin cancers in this study were from the plant using arsenic.

Burns and McDonnell prepared a report for the DOH in Missouri regarding the possible health effects of residents living near a creosote treatment plant.

Cancer incidence of the larynx, trachea, bronchus, and lung were greater among the nearby residents compared to both county and state rates.

Hospital discharge data showed higher rates of discharge for malignant neoplasms compared to the county. Neoplasms of the digestive, genital, and respiratory organs as well as diseases of the skin and some selected disorders of the digestive system were statistically higher than expected. Benign neoplasms, breast cancer and melanoma of the skin were all lower than expected compared with both state and county data.

Birth statistics were markedly worse for nearby residence compared to county and state data. Weight less than 2500 grams, gestation less than 37 weeks, and 5-minute Apgar score less than 8 (a measure of newborn health and responsiveness) were higher than both county and state rates. Total number of birth anomalies was higher in residents compared to state. Anomalies of the respiratory system, genital organs, poly/syndactyly, clubfoot, musculoskeletal, and of the skin hair, and nails were higher in residents compared to state rates.

The authors negate their findings in several ways. First, they note that there was no increase in genital organ cancer although this was one of the first reported cancers with a link to PAH exposure. This is true but the described exposure was prolonged dermal contact among chimney sweeps. As they point out early in the manuscript, this type of exposure is unlikely in the nearby residents. They also point out that the presumption is for county residents to be less exposed compared to nearby residents and state residents to have no appreciable exposure.

Eriksson and Karlsson investigate the risk factors for multiple myeloma in a case-control study of case-patients diagnosed with multiple myeloma in four counties of Sweden and reported to the Swedish Cancer Registry between July 1892 and June 1986. Exposure was assessed by questionnaire. This study also seems to focus on pesticide use and agriculture as the main exposures of interest. It appears that the targeted counties were those where agriculture abounded. The authors do report on creosote exposure (presumably coal tar creosote) but there were only 4 cases and 5 controls who were exposed. This yielded an odds ratio of 0.75 with a 90% confidence interval of 0.21 – 2.51.

Blair et al. report the findings of a population-based case-control study in 1993 examining risk factors for (non-Hodgkin's lymphoma) among white men in Iowa and Minnesota excluding large cities. This exclusion was made because the investigators were primarily concerned with determining whether and to what extent agricultural exposures contribute to the incidence of NHL. Information is obtained through

questionnaires on agricultural exposures, work history, medical conditions, and family history. Exposure was assessed by an industrial hygienist based on her understanding for potential occupational exposures likely to be associated with each job and/or industry. The authors note that their exposure assessment would likely result in "non-differential misclassification". This type of misclassification generally leads to bias toward the null value making it more difficult to detect an effect even if a causal relationship exists. As expected, very few significant findings were identified. However, there were increased risks reported among metalworking machinery industries, fabricated metal, transportation equipment, aircraft parts, toolmakers, misc. metal working and *paving occupations (3.4; 0.6 – 20.8)*. Cutting oils are identified as a potential risk factor. All these occupations and exposures share in common the potential for PAH exposure.

In 2002, Wong et al describe the results of their study of 1002 male and 55 female employees at 6 wood treatment plants in the U.S. The report was insufficient to fully evaluate the study design. However, there appeared to be several problems:

1) There appears to be no minimum requirement for time employment at the plant. Thirty-three percent of the cohort worked for less than 10 years; it is possible that a large percent worked for less than 1 year or 6 months, which will cause a dilution of the effect and lead to negative results.
2) Although the authors report the person-time contribution to the study, there is no assessment of the age distribution. The fact that only 10.6% of the sample is deceased suggests a very young sample.

3) The study is performed in two stages, a retrospective cohort mortality study and a nested case-control. The nested-case control study is fatally flawed. The choice of using mortalities from other causes as controls is a poor one. The measured relationship of the case to the exposure is confounded by this choice to the extent that the control death is associated with the exposure of interest.

Because of the lack of understanding of the health effects of coal tar creosote and the very small number of total deaths, this was an inappropriate control group. A better choice would have been to choose in a stratified random fashion anyone who belonged to the cohort and did not have lung cancer or multiple myeloma. This would have reduced the probability that the control suffered a death from a different cause but yet related to the same exposure as the case-patient.

That said, this study contributes to the sparse knowledge of the health effects of coal tar creosote. As one would expect in a worker cohort, the number of observed deaths was less than the number of expected deaths based on national rates. There were many rare cancers for which no observed cases were found. In most cases, where there was a deviation from expected numbers of deaths in either direction, the difference did not reach the level of significance. This is simply a reflection of the small numbers of deaths in this apparently young cohort of otherwise healthy workers. Certainly, a morbidity study of this cohort would have been more informative.

The study revealed increased rates of respiratory cancer and chronic endocardial disease in individuals occupationally exposed to creosote. It could be that certain individuals when exposed to coal tar creosote develop these two diseases early on in their exposure. However, even though the authors report significant decreases of mortality from lung cancer, endocardial disease and all heart disease, the mortality ratios are based on such small numbers, it becomes impossible to interpret the meaning or significance of this finding. The increased rates are interesting and deserve further attention.

The finding of multiple myeloma is particularly interesting in light of the nearly identical finding in the Flodin study. Unfortunately, any attempt to further understand the association in this cohort fails due to small numbers.

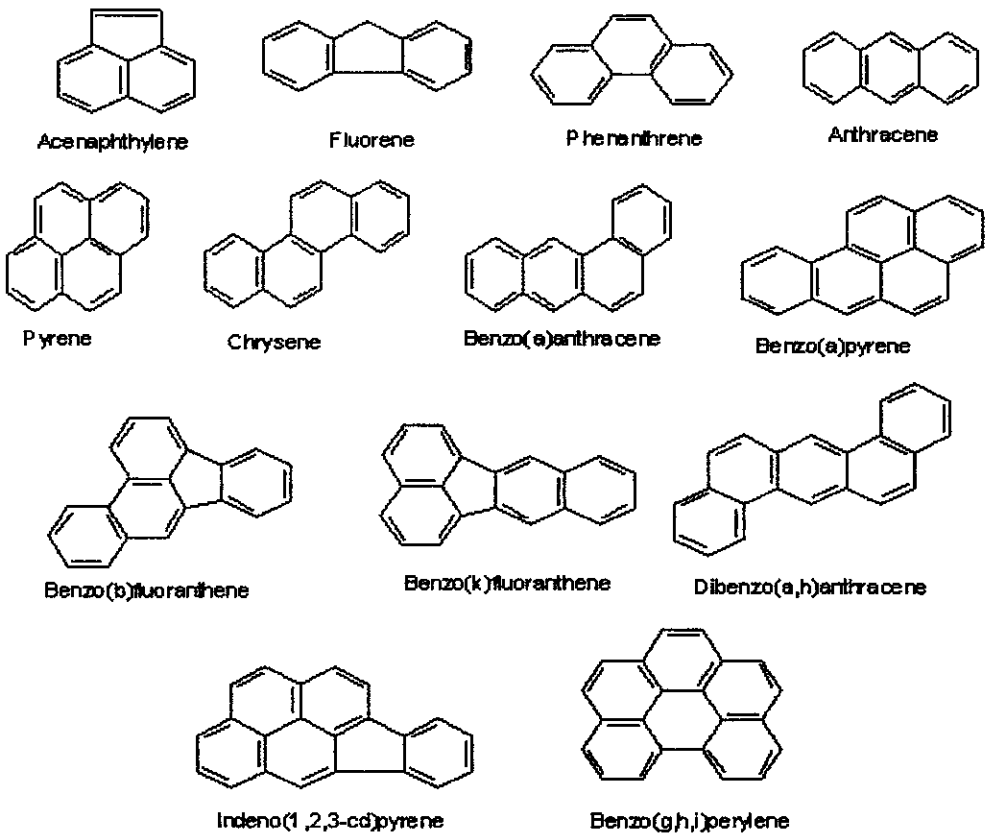
Other positive cancer findings with respect to creosote exposure include multiple myeloma[39,42] , Hodgkin's disease[40], non-Hodgkin's lymphoma.[40]

Polycyclic Aromatic Hydrocarbons

Definition and composition

Polycyclic aromatic hydrocarbons (PAHs) are a large group of diverse environmental organic pollutants, which consist of three or more aromatic rings that contain carbon and hydrogen only and share a pair of carbon atoms (Figure 4). These organic compounds are formed by the incomplete combustion of organic material like wood, coal or coke, coal tar and pitch, asphalt, and oil. The composition of the resultant PAH mixture is dependent on the source material, the temperature and other conditions of the burn. Much of the coal tar creosote mixture is comprised of PAHs.

Figure 4. Examples of Common PAH's.



Mechanism of action

PAHs cause damage to the DNA. They are metabolized to epoxides and then attach themselves to DNA. This attachment to DNA then cause errors in the cell's produciotn of proteins which then cause diseases such as cancer, birth defects and many other problems. PAHs have also previously been reported as both estrogenic and antiestrogenic depending on the experimental setting, today they are generally referred to as antiestrogens because they activate aryl hydorcaron receptor (AhR).[43] This can lead to suppression of estrogen response element (ERE). This mechanism is very similar to that of dioxin which will be discussed later.

Health effects

Percival Pott was the first to recognize and describe the health effects of PAHs from soot in the form of scrotum cancer in chimney sweeps.[44] His publication in 1775 sparked a widespread inquiry into environmental causes of cancer (Environmental and Occupational Medicine 1983). To date, many hundreds of studies have been published that identify and describe the carcinogenicity of a variety of chemicals classified as PAHs. Various forms of cancer have been associated with exposure to PAHs including scrotum,[45] bladder[46,47,48] and lung,[48,49,50,51,52] skin,[48,53] brain,[54,55] lymphatic[56] and hemotopoietic,[47,48,56] and pancreatic, [56] kidney, [57] stomach,[47,48,50] respiratory[42] and genitourinary systems, [50] and pharyngeal.[58] PAHs are generally recognized as genotoxic.[50] A general potency is displayed in Figure 5.

Breast Cancer

There is ample literature to support a causal association between breast cancer and PAH exposure. Since the 1985 Dusich study found increased rates of breast cancer in women exposed to creosote in their drinking water, there has been a flurry of research focusing on PAHs and breast cancer.[59, 36] Rundle et. al. published the results of a case-control study examining PAH levels in women with breast cancer and women with benign breast disease and benign breast disease with hyperplasia.[60] When adduct levels in the tumors were dichotomized and then compared to those in benign tissue, the odds ratio was 5.26, p-value 0.03. In addition, the adducts from tumor tissue and normal tissue in the cases were highly correlated however, there was no relationship between smoking status and adduct levels. The authors offer two explanations for their findings: 1) cases could be more sensitive to the carcinogenic effects of environmental PAH. Hence, the combination of exposure and susceptibility caused the breast cancer; or, 2) changes in the tumor cells lead to greater formation and accumulation of adducts. Thus, the findings may indicate PAH-DNA adducts play a role in the further progression of malignant cells. Recently, Pliskova et al. report the findings of laboratory testing where she and others examined the effects of several PAHs two of which were benz[a]anthracene and benzo[a]pyrene. They found that these two chemicals promoted human breast cancer.[43]

Reproductive Health Effects

Animal studies have shown that mice fed high levels of PAH during pregnancy had difficulty reproducing. The effect was also seen in their offspring.[61,62] PAHs have

produced liver, lung, lymphatic tissues, and nervous system cancers in the offspring of rodents.[63,64,65] In human offspring, Perera et al report smaller birth size among newborns with PAH-DNA adducts above the median ($3.85/10^8$ nucleotides).[66] She explains that exposure to PAHs can occur from inhalation of polluted air and from diet including grilled or smoked foods and vegetables grown in contaminated areas. Although animal data suggests a 10-fold decrease in fetal PAH exposure compared to maternal exposure,[67,68] Perera et al found higher PAH-DNA adduct levels in the newborns compared to their mothers a finding that is later confirmed by Whyatt et al.[69] This finding suggests that although the fetus incurs a reduced level of exposure, the young cells have a decreased efficiency for detoxification and/or DNA repair. Birth weight, length and head circumference were all statistically significantly lower in newborns with high (above the median) leukocyte levels of PAH-DNA adducts compared to those with low (below or equal to the median) levels. This analysis controlled for maternal height, age, education level, history of low birth weight, maternal alcohol consumption, gestational age, newborn's gender and plasma cotinine. Of particular interest was the fact that head circumference was inversely related to PAH-DNA adducts both before and after birth weight was included in the model suggesting asymmetrical growth retardation. The authors note that reduced head circumference of 1 – 2 centimeters in newborns has been associated with reduced mental and psychomotor development in early childhood[70,71] and that head circumference has been correlated with brain size, intelligence quotient, and cognitive function.[72,73]

Table 1: Studies that examine the cancer effects of coal tar creosote

First Author	Year	Study Design	Health Effect	Exposure Measure	Effect Measure	Significance /CI
Dusich	1980	SMR[i] study	Breast Cancer	Residents who drank creosote contaminated water near a creosote plant	E = 113 [c] U = 78	0.0005 [d]
			Gastrointestinal CA		N/A	0.05 [d]
Flodin	1987	Case-control (referent[m])	Multiple myeloma	Questionnaire	4.7 [e]	1.02 – 18.0 [b]
Dean	1988	Case-control (matched)	Breast Cancer	Questionnaire	Obs =113/100,000 Exp =115/100,000	N/A
Persson	1989	Case-control (referent[m])	Hodgkin's disease	Questionnaire	10.7 [f]	1.1 – 103 [g]
			Non-Hodgkin's lymphoma		9.4 [f]	1.2 – 69 [g]
Karlehagen	1992	SMR[i] study	Lip Cancer	Creosote workers	2.50[a]	0.81 – 5.83 [b]
			Skin Cancer		2.37 [a]	1.08 – 4.50 [b]
			Malignant lymphoma		1.9 [a]	0.83 – 3.78 [b]
Burns	1992	Community comparison and SMR study	All Health Effects	Residents near Creosote Plant	N/A	N/A
Eriksson	1992	Case-control	Multiple myeloma	Questionnaire	0.75	0.21 – 2.51 [g]

Blair	1993	Case-control	Non-Hodgkin's lymphoma	Asbestos and creosote exposure	1.0 [h]	0.7 – 1.5 [b]
				Paving occupations	3.4	0.6 – 20.8
Wong	2002	Retrospective cohort SMR[j] study	Multiple myeloma	Creosote workers	606.1[j]	165.2 – 1522.0 [b]
			Chronic endocardial disease [k]		1744.9[j]	< 0.01 [d]
			Cancer of the repiratory system [k]		354.6[J]	N/A
Dahlgren	2003	Cross-sectional community comparison	All cancer rate	Residents near a creosote plant	4.8[l]	< 0.05 [d]
Brender	2003	Cross-sectional community comparison	Difficulty becoming pregnant	Residents near a creosote plant	3.3	1.3 – 8.7 [b]
			Chronic bronchitis		2.7	1.3 – 5.6 [b]

[a]Standardized incidence ratio (SIR)

[b]95% confidence interval

[c] E = exposed cases/100,000 population; U = unexposed cases/100,000 population

[d]p-value

N/A = information not published

[e] Mantel-Haenszel rate ratio controlling for age exposure to fresh wood, exhaust, concrete- and brickwork, sulfonylurea, gamma radiation, ex-smoking, farmers and gender. Crude rate was 6.0.

[f] Adjusted odds ratio controlling for age at diagnosis, gender, farming and exposure to fresh wood as well as any other exposures with at least a doubled risk for HD or NHL.

[g]90% confidence interval

[h] Adjusted odds ratio

[i]Standardized morbidity ratio

[j]Standardized mortality ratio where 100 = the null value

[k] In workers with less than 10 years of employment

[l] Rate ratio

[m]The same controls were used for both the Persson and Flodin studies

PAH-DNA adducts:

Environmental exposures can be estimated by measuring concentrations of pollutants in air, water, food, or wipe tests. When such measurements are obtained in a scientific manner, the environmental exposure results can be used to estimate individual exposures. PAHs leave characteristic "fingerprints" when they bind to nucleotides in DNA to form chemical-DNA adducts (Figure 5). If unrepaired prior to cell replication, the adduct can cause permanent gene alteration and expression resulting in cell mutation that increases the probability of cancer development (Figure 6).[74] PAH-DNA adducts are the net result of exposure, absorption, activation, detoxification, and repair and are commonly used to measure the biologically effective dose of PAH.[66,74] The literature is rich with studies on smoking-related DNA adducts in tissue and protein adducts in blood.[75] Many of these studies have paved the way for a better understanding of environmental exposures to pollutants. Poirier and Weston review the state of the art of adduct testing.[76] PAH-DNA adduct levels in white blood cells reflect environmental exposure to PAHs.[77,78] Therefore, another approach to determining the personal exposures and corresponding health effects of creosote exposure is to measure the PAH-DNA adducts.

Figure 5. Activation of PAH compounds

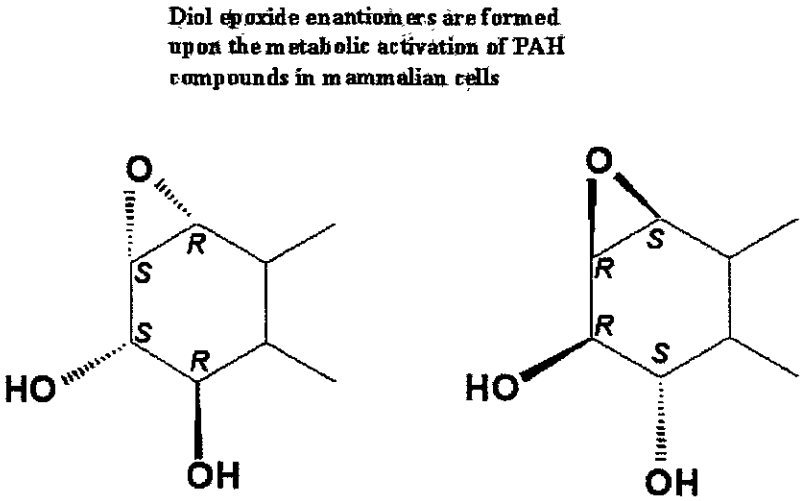
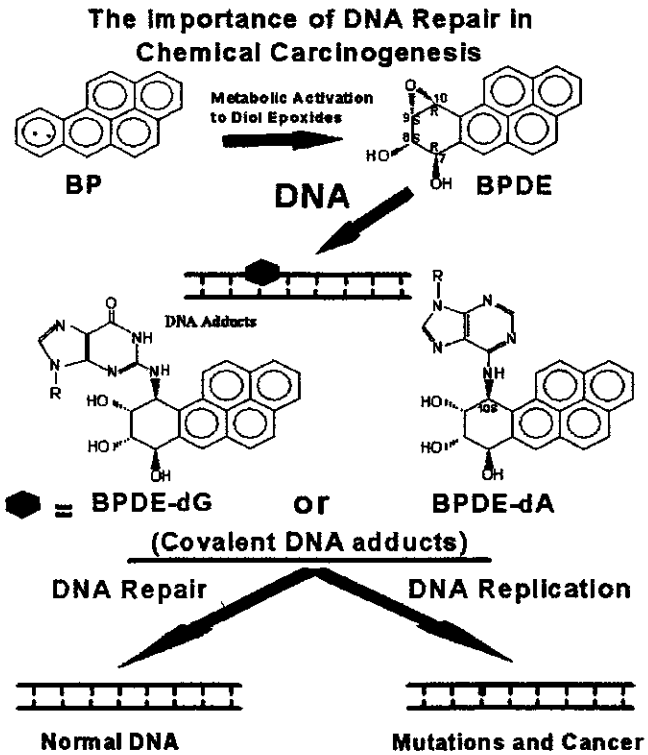


Figure 6. PAH-DNA Adducts and Chemical Carcinogenesis.



Residents of the small, rural community in Grenada who were located next to a wood processing plant were tested for the presence of PAH-DNA adducts. Subjects selected for biomonitoring were randomly chosen from a total of 103 total residents who are a part of an ongoing litigation against the wood treatment plant (Koppers) due to their concern about associated health problems. Inclusion criteria for the randomly selected subjects were: (1) above 20 years old, and (2) living in the same residence for at least 5 years. Exclusion criteria consisted of the fact that (1) none of the exposed group members were employed at the wood treatment plant. Twenty-nine subjects were selected at random in total.

Testing for PAH-DNA Adducts was performed on 24 of the 29 randomly selected subjects. Five (5) subjects failed to appear to have their blood drawn for the adduct testing. A comparable control group for comparison with our study group for PAH-DNA adducts was located in a town in Florida. Laboratory personnel who preformed the testing were blinded as to the exposure status of the test samples.

Whole blood was collected in Vacutainer CPT tubes (Becton Dickinson) from 24 of the 29 subjects. These tubes are Ficoll-containing tubes that are drawn under vacuum. The tubes were then transported to a laboratory for centrifugation and separation (by Ficoll Method) of mononuclear cells within 24 hours. The samples were kept on dry ice and sent for DNA adduct analysis to England. PAH-DNA adducts were measured utilizing the ^{32}P-post labeling technique.[78] The PAH-DNA adduct levels have been shown to quantify PAH exposure[75] and has been used in several other small studies[59].

Twenty-four (24) of the 29 subjects, who were randomly identified, volunteered for PAH-DNA Adduct testing. Seven unexposed subjects were used for comparison. The demographics of the two groups are shown in Table 2. The mean PAH-DNA adducts in the comparison group was 0.75 per 10^8 nucleotides (range = 0.54 – 0.99 per 10^8 nucleotides) whereas the exposed subjects had a mean adduct level of 4.11 per 10^8 nucleotides (range = 1.72 – 8.52 per 10^8 nucleotides). The exposed group has 5.48 times higher PAH-DNA adduct levels than the comparison group. We did not adjust for dietary confounders (BBQ intake) as that history was unavailable at the time in our comparison group.

Regression analysis shows that even after controlling for race/ethnicity, age, gender and smoking status, exposed persons have a statistically significantly higher PAH adduct level compared to unexposed. For every unit of increase in PAH adduct level in the unexposed group, the exposed group increased its level by 3.58 units ($p = 0.0006$) (Table 3).

These findings of PAH-DNA adducts on residents living near a creosote wood treatment plant reflect environmental exposure to PAHs.[75, 77] The residents have a mean level of PAH-DNA adducts 4.7 times higher than that of our control population (Figure 7). These results indicate that the residents are being exposed to higher levels of PAH contaminants than the controls. Transformed or activated PAHs can bind to DNA forming adducts, which is widely believed to be the initiating step in chemical carcinogenesis.[79,80] Few

studies have attempted to explain a dose-response relationship between environmental exposure to PAHs and PAH-DNA adduct levels. Higher PAH-DNA adduct levels predict a higher risk of cancer.[81] In addition, studies in animals demonstrate that the levels of DNA adducts are related to PAH contaminated sites as compared to reference sites.

Table 2. Demographics and PAH-DNA Adduct Results in residents & comparison group.

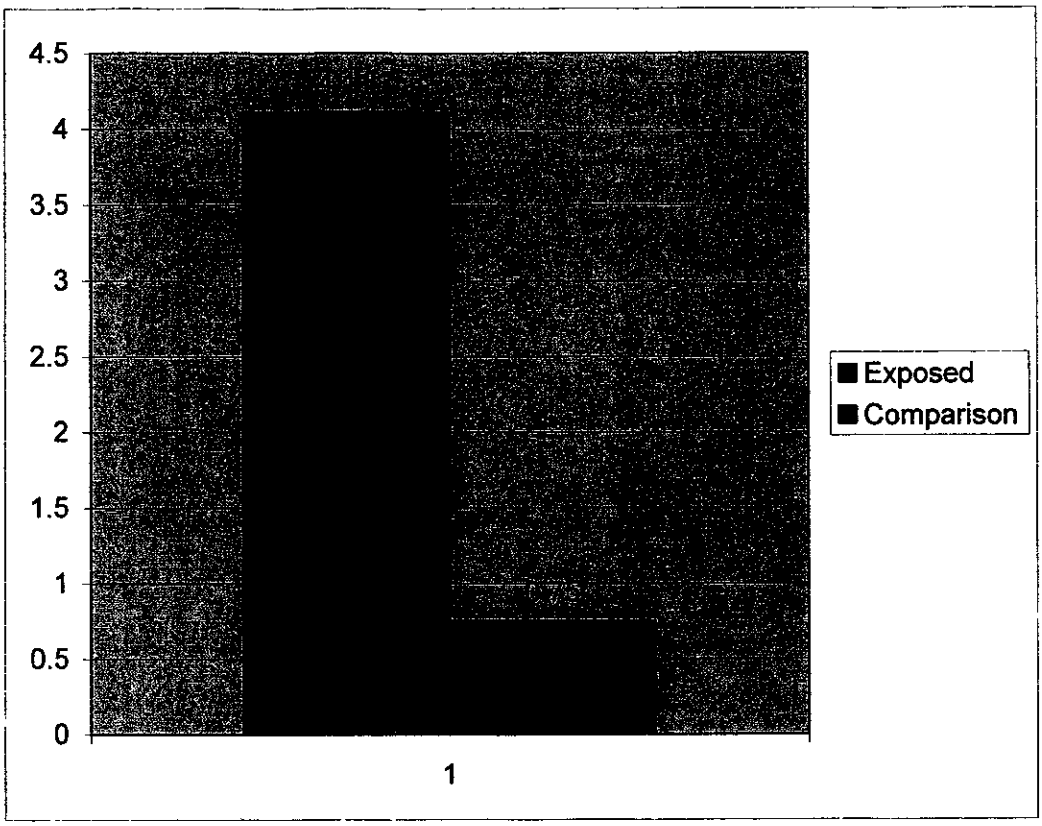
ID	Ethnicitiy	Age	Sex	Smoking Status	Average number of adducts per 10^8 Nucleotides	S.D.	Population
0001	Black	44	female	non smoker	1.72	0.02	Exposed
0007	Black	22	female	non smoker	3.61	0.21	Exposed
0026	Black	42	male	non smoker	5.91	0.54	Exposed
0028	Black	38	female	non smoker	2.86	1.04	Exposed
0030	Black	51	female	smoker	2.91	0.41	Exposed
0037	Black	38	female	smoker	2.74	0.13	Exposed
0039	Black	49	female	smoker	5.44	0.13	Exposed
0047	Black	70	male	smoker	3.86	0.97	Exposed
0050	Black	22	female	non smoker	5.05	1.27	Exposed
0053	White	73	female	non smoker	7.06	0.80	Exposed
0056	White	54	female	non smoker	5.16	0.08	Exposed
0063	Black	48	male	smoker	2.85	0.77	Exposed
0065	White	55	male	non smoker	6.51	0.00	Exposed
0067	White	42	male	ex smoker	4.83	1.43	Exposed
0069	Black	30	female	non smoker	4.33	0.93	Exposed

0076	Black	60	female	non smoker	2.95	0.76	Exposed
0081	White	54	male	smoker	3.72	0.93	Exposed
0082	White	55	female	smoker	2.13	0.12	Exposed
0088	Black	72	male	smoker	3.29	0.94	Exposed
0091	N.A.	21	female	non smoker	4.89	0.84	Exposed
0098	Black	73	female	non smoker	2.49	0.44	Exposed
0202	Black	90	male	non smoker	8.52	1.36	Exposed
0214	White	39	male	non smoker	3.18	0.41	Exposed
0215	Black	78	male	non smoker	2.62	0.20	Exposed
225 855	White	26	female	non smoker	0.58	0.08	Comparison
225 856	White	45	female	non smoker	0.54	0.12	Comparison
225 858	N.A.	N.A.	female	N.A.	0.77	0.07	Comparison
225 859	White	32	male	non smoker	0.99	0.03	Comparison
225 860	White	49	male	ex smoker	0.97	0.31	Comparison
225 862	White	28	female	non smoker	0.80	0.21	Comparison
225 864	White	33	female	smoker	0.58	0.10	Comparison

Table 3: Multiple linear regression comparing PAH adduct levels among exposed and unexposed persons controlling for race/ethnicity, age, gender and smoking status.

	Exposed	Unexposed	Slope β	p-value
Race/ethnicity				
White	9 (32.14%)	6 (100%)		
Black	19 (57.86%)	0		
Missing	1	1		
Mean age (SD)	48.79 (8.86)	35.5 (9.35)		
Gender				
Male	11 (37.93%)	2 (28.57%)		
Smoking status				
No	21 (72.41)	5 (83.34%)		
Yes	8 (27.59%)	1 (16.67%)		
Mean adduct level (SD)	**4.11 (1.7)**	**0.75 (0.19)**	**3.58**	**0.0006**

Figure 7. DNA PAH adduct levels adduct levels of residents and comparison group



Dioxins:

Definition and composition

What is referred to as dioxin actually represents a class of chemicals named chlorinated dibenzo-p-dioxins (CDDs). They are a family of 75 different compounds also commonly called polychlorinated dioxins. These compounds have harmful effects. The CDD family is divided into eight groups of chemicals based on the number of chlorine atoms in the compound.

1. mono-chlorinated dioxin(s).
2. di-chlorinated dioxin (DCDD)
3. tri-chlorinated dioxin (TrCDD)
4. tetra-chlorinated dioxin (TCDD)
5. penta-chlorinated dioxin (PeCDD)
6. hexa-chlorinated dioxin (HxCDD)
7. hepta-chlorinated dioxin (HpCDD)
8. octa-chlorinated dioxin (OCDD).

The chlorine atoms can be attached to the dioxin molecule at any one of eight positions (Figure 8). The name of each CDD indicates both the number and the positions of the chlorine atoms. For example, the CDD with four chlorine atoms at positions 2, 3, 7, and 8 on the dioxin molecule is called 2,3,7,8-tetrachlorodibenzo-p-dioxin or 2,3,7,8-TCDD. 2,3,7,8-TCDD is one of the most toxic of the CDDs to mammals and has received the most attention. Thus, 2,3,7,8-TCDD serves as a prototype for the CDDs. CDDs with toxic properties similar to 2,3,7,8-TCDD are called "dioxin-like" compounds. There is a

closely related chemical which also has dioxin-like properties which are identical to dioxins except there is only one oxygen between the two benzene rings as shown in the figure one (Figure 9). Polychloronatated Biphenyls (PCBs) also have dioxin like toxicity and exert a similar toxic effect.

Figure 8. Examples of Dioxins and furans.

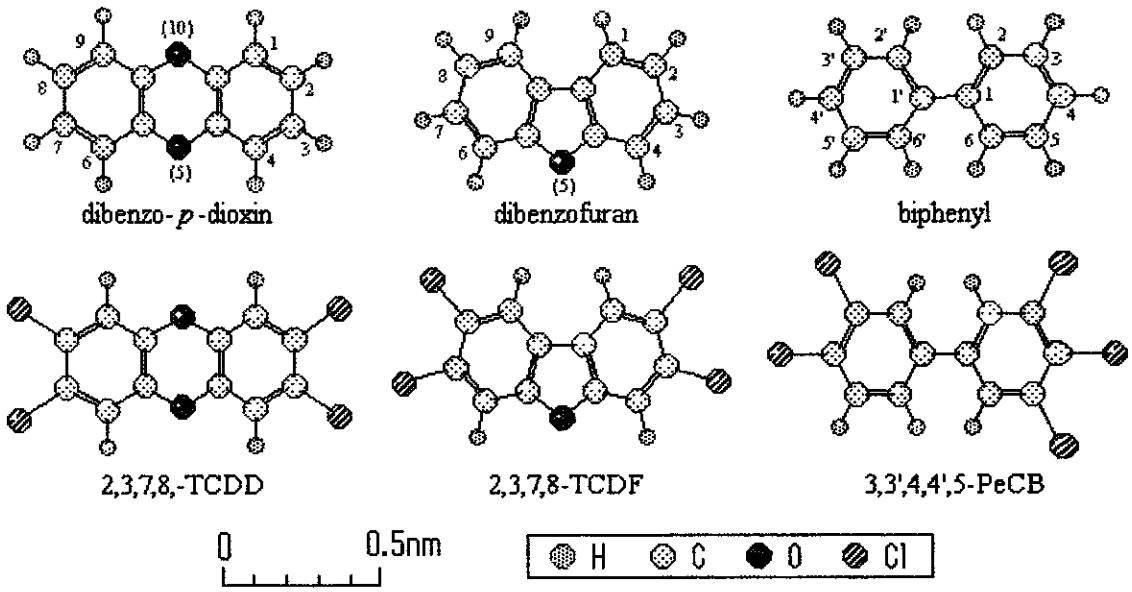
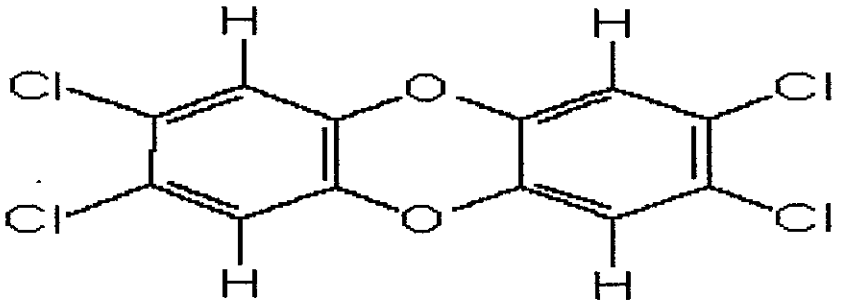
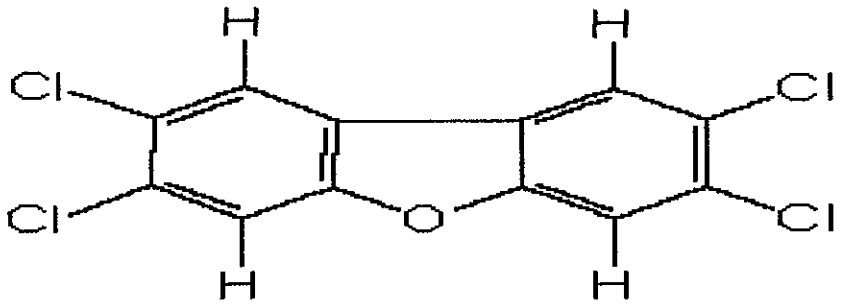


Figure 9. Image from ATSDR: Chemical Structure of Dioxins vs Furans.



2,3,7,8-TCDD



2,3,7,8-TCDF

Toxic risk assessment

The various dioxins and furans have different levels of toxicity depending on how toxic they are in various test systems. The toxic equivalency (TEF or TEQ) of a dioxin or furan is a measure of how toxic it is relative to 2,3,7,8 TCDD (Please refer to TEF / TEQ section). An organochlorine with a TEQ of .05 is 5% as poisonous as 2,3,7,8 TCDD. Table 4 is a listing of the TEF for the various dioxins, furans and the closely related dioxin like PCBs.

One problem with the use of TEQs for risk assessment in this case is the fact that the PAHs also stimulate the Ah receptor. These Ah receptor agonists elicit responses in humans that are consistent with a receptor-mediated pathway, but the combination of these two classes of Ah receptors is unstudied. Therefore, a TEF/TEQ approach based solely on intake of dioxins does not take into account the additional PAH exposure that increases the responses associated with persistent low-level occupation of the Ah receptor by dioxins and PAHs together.[82]

Table 4. Table from ATSDR Toxicological Profile for Dioxins

Table 2-11. Toxicity Equivalency Factors (TEFs) for Halogenated Hydrocarbons

CDFs	EPA current recommended values[a]	CDDs	EPA current recommended values[a]	PCBs	WHO/IPCS interim value[b]
monoCDFs	0	monoCDDs	0	3,3'4,4'-tetraCB	0.0005
diCDFs	0	diCDDs	0	3,3',4,4',5-pentaCB	0.1
triCDFs	0	triCDDs	0	2,3,3',4,4'-pentaCB	0.0001
2,3,7,8-tetraCDF	0.1	2,3,7,8-TCDD	1	2,3,4,4',5-pentaCB	0.0005
other tetraCDFs	0	other tetraCDDs	0	2,3',4,4',5-pentaCB	0.0001
1,2,3,7,8-pentaCDF	0.05	2,3,7,8-pentaCDD[c]	0.5	2',3,4,4',5-pentaCB	0.0001
2,3,4,7,8-pentaCDF	0.5	other pentaCDDs	0	3,3',4,4',5,5'-hexaCB	0.01
other pentaCDFs	0			2,3,3',4,4',5-hexaCB	0.0005
2,3,7,8-hexaCDF[c]	0.1	2,3,7,8-hexaCDD[c]	0.1	2,3,3',4,4',5'-hexaCB	0.0005
other hexaCDFs	0	other hexaCDDs	0	2,3',4,4',5,5'-hexaCB	0.00001
2,3,7,8-heptaCDF[c]	0.01	2,3,7,8-heptaCDD[c]	0.01	2,3,3',4,4',5,5'-heptaCB	0.0001
other heptaCDFs	0	other heptaCDDs	0	2,2',3,3',4,4',5,-heptaCB	0.0001
octaCDF	0.001	octaCDD	0.001	2,2',3,4,4',5,5'-heptaCB	0.00001

[a]Derived from EPA 1989e
[b]Derived from Ahlborg et al. 1994
[c]Any isomer that contains chlorine in the 2,3,7,8-positions

CDDs = chlorinated dibenzo-*p*-dioxins; CDFs = chlorinated dibenzofurans; PCBs = polychlorinated biphenyls; TCDD = tetrachlorodibenzo-*p*-dioxin

Environmental availability and routes of exposure

Dioxins are widely encountered toxic substances. They are ubiquitus in the environment, are persistent and, being fat soluble, they tend to accumulate in the fatty tissue of higher animals - including humans. Their resistance to degradation and semi-volatility increases the likelihood that they will be transported over long distances. In addition, dioxins which were released into the environment many years ago are still contributing to current exposure. Even very small dioxin concentrations are known to cause negative environmental and human health effects. Those at greatest risk are usually the most vulnerable among us, like children. In 2000 the USEPA called for a reduction in environmental dioxin because the level of contamination was too close to the lowest dose known to create an adverse health effects.[83]

In this case there is a major source of dioxin from Koppers which has caused the levels of dioxins to be significantly elevated in the resident's house dust, soil and blood compared to control vales in unexposed communities. These nearby residents of the wood treatment plant have significantly increased levels of hepta and octa dioxins in their environmental samples and correspondingly in their blood.

Dioxins are mainly produced as unwanted by-products of industrial processes. In this case we see the high levels of dioxins in this neighborhood because of the use of pentachlorophenol (PCP) used to preserve wood along with creosote (refer to the section of this report on PCPs). PCP contains an unwanted byproduct of the more highly chlorinated CDDs (those with more chlorine atoms), like hepta (HpCDD) and octa

(OCDD) dioxins but 2,3,7,8-TCDD is not usually found. The use of PCP has been restricted to certain manufacturing applications because of its high level of toxicity.

The background daily intake of dioxins and dioxin-like compounds is still above the levels recommended by the World Health Organisation (WHO), despite the fact that dioxin levels have been decreasing in the recent years in all countries for which data for the last 10 to 15 years are available. On average, exposure fell by 10% per year between the mid-eighties and the mid-nineties.

Health effects of dioxin

Diseases, which have been linked to dioxin, seem endless. Research has shown that it causes skin disfiguration, Hodgkin's disease, non-Hodgkin's lymphoma, soft-tissue sarcoma, leukemia, liver cancer, and porphyria cutanea tarda (PCT, a metabolic disorder).

TCDD has been classified as a known human carcinogen by the International Agency for Research on Cancer.[84] Epidemiological data have shown increases in soft-tissue sarcomas, respiratory system tumors and all cancers combined.[85] TCDD is a carcinogen in all species and strains of laboratory animals tested (e.g., mice, rats, hamsters) with tumors detected in the stomach,[86] liver, thyroid, respiratory tract, and other organs and tissues. Long-term rodent carcinogenicity studies have shown that TCDD is a potent carcinogen, with the most seriously affected organ being liver in female rodents.[87,88,89]

Ingesting dioxin can also result in congenital malformations, spontaneous abortions, and a slow wasting syndrome followed by death similar to the AIDS syndrome. Dioxin exposure causes damage to the urinary and hematological systems, growths in the colon,

gall bladder, multiple myeloma, and lung, larynx and prostate cancer. Kovevinas reviewed the literature on the human health effects of dioxins and found increased cancer risk reports for all cancers combined; lymphatic and haematopoietic systems; hepatobiliary cancer in women; lung and rectal cancer in men; breast cancer; endometrial cancer, and testicular cancer.[90] With the noted exception of chloracne and increased liver enzymes other non-cancer health effects were not consistently proven. Health effects that were implicated but not proven were cardiovascular diseases, diabetes, reproductive problems, thyroid function, neurological/psychological effects and respiratory system anomalies.

According to ATSDR, a government agency, dioxin's health effects include endocrine disruption, reproductive impairment, infertility, birth defects, lowered sperm counts, impaired neurological development, damage to the kidneys, and metabolic dysfunction. There is no evidence that there is a safe level of dioxin exposure below which none of these effects will occur, although as a practical matter we do not know what the No Adverse Effect Level is for humans.

Dental problems and Dioxin

Rodent studies have shown dioxin-related dental anomalies such as discoloration, gum deformation, missing teeth and smaller teeth .[91,92,93] One study also discusses a corresponding impairment of normal skull formation in exposed rats.[91] In addition, several human epidemiologic studies have produced similar results. One study reports the dental effects in children exposed to the environmental pollution from a wood preservative manufacturing plant. The plant manufactured a wood preservative that

contained a mixture of PCPs. Dioxin occurred as an unintended byproduct. Mothers were exposed and the toxic material concentrated in the breast milk that was subsequently delivered to their infants. The study found increased rates of developmental defects in first molars and hypomineralized teeth in children whose mothers lived downstream on the polluted river.[94] Wang et al. report a dose-response effect of increasing dental defects with increasing maternal serum PCB levels, childhood PCB and PCDF levels and duration of breastfeeding.[95] The authors reported dental defects including neonatal development of teeth, greater number of permanent teeth prior to age 11 and fewer permanent teeth after the age of 10. Jan and Vrbic showed dramatic effects of cavities, abnormal enamel in permanent teeth.[96] In appendices, I provide the pages from the ATSDR that outline some of the health effects of dioxin. These appendices give exhaustive detail on how damaging dioxins can be. It must be kept in mind that there have not been studies that looked at the combined effects of PAHs and dioxins but is very clear that both exert a very damaging effect on the fetus. Both PAHs and dioxins cause cancer, prematurity, low birth weight for gestational age, birth defects, altered sexual development, immune system damage. They both stimulate the Ah receptor, which is one of the main mechanisms that cause health damage. The residents living near the Koppers plant are exposed to elevated levels of both dioxins and PAHs. This combined exposure is why they are experiencing such high rates of cancer, developmental problems, respiratory effects, neurological injury, autoimmune diseases and other illnesses.

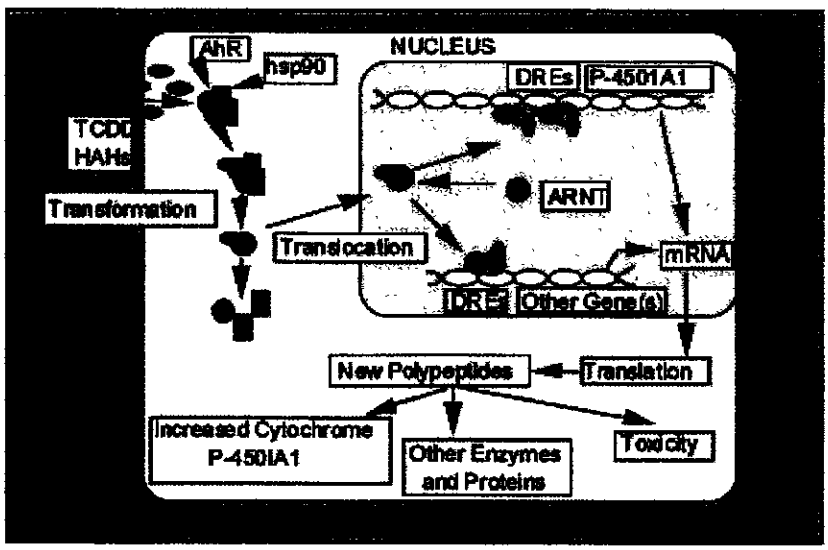
Mechanism of action

Why does dioxin have so many different effects? And why do some diseases show up in some investigations but not others? Dioxin is an organochlorine, or a man-made

chemical, which has chlorine bound to organic compounds. Organochlorines are foreign to the biology of life. They are not created naturally in the bodies of humans or other terrestrial animals. It is a requirement of all living things to be able to break down or biodegrade chemicals that enter our bodies. Organochlorines are extremely stable, sometimes taking hundreds of years to break down. Our bodies do not have efficient mechanism to break down or metabolize things like dioxin.

These compounds bind to an intracellular receptor called the aryl hydrocarbon receptor (Ah Receptor) and activate the receptor. The dioxin-Ah Receptor complex then travels to the nucleus of the cell and binds to specific sequences in the DNA called dioxin responsive elements (DRE). The binding of the dioxin-Ah Receptor complex to the DRE causes the expression of the associated genes to be altered. It is this alteration in gene expression that causes the toxic effects that are observed (Figure 10).

Figure 10. Dioxin-Ah receptor complex sequence.



When dioxin is taken into the cell nucleus, it works like a "turn on" valve, which begins the production of various enzymes. Enzymes cause a vast amount of bodily activity. The huge number of enzymes does two things: start and stop biological activity. Much activity is stopped by the enzyme *biodegrading*. But the organochlorines degrade very slowly (estimated half-life of 7 to 12 years). Thus, dioxin functions like a permanent "on" switch that keeps a particular bodily process going.

Many toxic chemicals are linked with a specific illness, such as lead and brain damage or asbestos and mesothelioma. Others are linked with several illnesses. Dioxin is tied to such a very large number of diseases because it is a *cancer-enhancer*. Dioxins intensify (promote) cancers, which other toxics begin.

As Barry Commoner explains, "...dioxin greatly enhances the activity of the enzyme system that converts most environmental carcinogens into active agents. Apparently, dioxin can so powerfully stimulate the enzyme as to sharply increase the activity of the small amounts of carcinogens present in food, water, and air and thereby intensify their effect on tumor incidence. In effect, dioxin influences tumor production by enhancing the activity of carcinogens..." The enzyme which dioxin induces is aryl hydroxylase (AhH). It is the enzyme which adds an oxygen to cancer causing molecules like benzo(a)pyrene which is one of the PAHs present in creosote.

This is why the dioxin exposure experienced by the residents near the Koppers Plant is so dangerous because it greatly magnifies the toxicity of chemicals that are also present in increased amounts in the homes and blood of the residents near the Koppers Wood Treatment Plant.

This ability to magnify toxic effects is why dioxin has different effects on different people. If a group of workers has already been exposed to chemicals, which cause Hodgkins disease, dioxin will speed up the process and research shows that they have an increased rate of Hodgkin's disease. If a community has been exposed to chemicals, which cause leukemia, dioxin will increase the rate of leukemia.

On the other hand, if dioxin is spread on a group of people who have come into contact with a variety of pollutants (because they have different kinds of jobs), then there would be small increases in many diseases. The only way researchers could measure the effects would be to add a long list of diseases and compare their total frequency in a group which was exposed and another group which was not exposed. This is similar to what Marilyn Fingerhut and other researchers did when they found that combined cancers had a 46% higher likelihood of occurring amongst workers who had been exposed for only one year (and after a 20 year "latency" period had elapsed).

Another problem with dioxin research is finding a group of "unexposed" people to use for comparison. There used to be virtually no dioxin in human tissue. This changed during the 1930s, when industry began producing large amounts of organochlorines such as insecticides, fungicides and chemical fertilizers.

To get an idea of how toxic dioxin can be we can refer to an explosion in Seveso, Italy in 1976. This accident was a major disaster when an explosion spewed 1 to 4 pounds of dioxin into the air over the town. Studies are now showing a broad range of cancers in the people. Very small amounts of dioxin are very dangerous.

Developmental effects have been shown to occur at extremely low levels in monkey offspring studies. In these studies 2,3,7,8-TCDD was administered chronically to rhesus monkeys. Decreased offspring survival was found when mothers were exposed continuously during pregnancy to 6.4×10^{-4} µg/kg/day in the feed.[97] In addition, alterations in peer-group behavior[97,98] and cognitive deficits were observed in the offspring of rhesus monkeys exposed to 1.2×10^{-4} µg/kg/day in the diet for 7 months prior to mating and during mating and lactation (16 months total duration). Significant alterations were observed in play behavior, displacement, and self directed behavior. Exposed monkeys tended to initiate more rough-tumble play bouts and retreated less from play bouts than controls, were less often displaced from preferred positions in the playroom than the controls, and engaged in more self-directed behavior than controls. Cognitive function was altered as evidenced by impaired-reversal-learning performance in the absence of impaired delayed-spatial-alterations performance;[97,99] The No Observable Adverse Effect Level (NOAEL) was not identified for these effects. Schantz et al. also found increased and prolonged maternal care of these infants.[100]

The Lowest Observable Adverse Effect Level (LOAEL) of 1.2×10^{-4} µg/kg/day identified for neurobehavioral effects identified in the Schantz et al. (1992) study was used by ATSDR to derive a chronic oral Minimum Risk Level (MRL) of 1×10-6 µg/kg/day. Minimum Risk Levels are set by ATSDR based on studies usually animal studies of one chemical at time. They set these levels so there can be quantitative values for purposes of assessing environmental exposures.

This level of 1×10-6 µg/kg/day of dioxin is the lowest MRL of any chemical. It means that the level of dioxin exposure that is thought to be safe for a 22 pound (10 Kg) 2 year old human to ingest per day is 10 picograms. A picogram is one quintillionth of a gram. There are 454 grams in a pound. The picogram is 0.000000000000001 grams. This is a small quantity and is almost assuredly exceeded in the case of the children who lived near the Koppers Plant.

In fact ATSDR used a different uncertainty factor than they usually use to reach this level. The actual MRL should be 3 times lower or 3 X 10^{-7} Ug/kg/ day. The reason for this inconsistency is not provided by ATSDR in it Profile.

Table 5 compares the adjusted exposed means with that of a comparison group. All of the dioxins in the exposed group in Grenada were statistically significantly. The statistically significant increases in the dioxins are similar to another creosote resident population we studied.[101]

Table 5. Dioxin Levels are significantly higher for Exposed Residents than for Controls.

Type of Dioxin	Exposed adjusted mean dioxin[a]	Unexposed mean dioxin[a]	P-value for the difference
2,3,7,8-TCDD	3.03	**	N/A
1,2,3,7,8-PeCDD	7.64	6.25	0.08
1,2,3,4,7,8-HxCDD	9.62	5.19	0.006
1,2,3,6,7,8-HxCDD	46.7	34.3	0.06
1,2,3,7,8,9-HxCDD	9.44	6.5	0.009
1,2,3,4,6,7,8-HpCDD	123.21	50.3	0.001
OCDD	954.4	347.5	0.0005
1,2,3,7,8-PeCDF	0	1.045	**
2,3,4,7,8-PeCDF[b]	3.33	5.85	<0.0001
1,2,3,4,7,8-HxCDF	7.27	5.035	0.01
1,2,3,6,7,8-HxCDF	4.37	4.88	0.29
1,2,3,7,8,9-HxCDF	0	**	N/A
2,3,4,6,7,8-HxCDF[b]	0.993	2.03	<0.0001
1,2,3,4,6,7,8-HpCDF	0.99	**	N/A
1,2,3,4,7,8,9-HpCDF	0	0.907	**
TEQ	31.43	15.35	<0.0001

[a]Adjusted for age, gender, race/ethnicity and smoking status (yes/no)

[b] Significantly lower in the exposed group

** Values were not available or were non-detect.

TOXICITY EQUIVALENT FACTOR (TEFs/TEQs):

Dioxins

Toxic Equivalents (TEQ) is used to report the *toxicity-weighted masses* of mixtures of dioxins. The TEQ method of reporting is more significant than simply reporting the *total number of grams* of a mixture of toxic compounds since the TEQ method offers toxicity information about the mixture.

In the TEQ method each dioxin compound is assigned a Toxic Equivalency Factor, or TEF (see the tables below). This TEF denotes a compound's toxicity relative to 2,3,7,8-TCDD, which is assigned the maximum toxicity designation of one. Other dioxin compounds are values related to its toxicity relative to that of 2,3,7,8-TCDD. The World Health Organization (WHO) developed this system.

Since chemicals often occur in mixtures, rather than as pure compounds, the TEF values are added together to give a Toxicity Equivalence (TEQ) for the whole mixture. Therefore, the TEQ is the amount of TCDD it would take to equal the combined toxicity of the whole mixture.

PAHs

"EPA has recommended that a toxicity equivalency factor (TEF) be used to convert concentrations of carcinogenic polycyclic aromatic hydrocarbons (cPAHs) to an equivalent concentration of benzo(a)pyrene when assessing the risks posed by these substances. These TEFs are based on the potency of each compound relative to that of

benzo(a)pyrene. For the toxicity value database, these TEFs have been applied to the toxicity values.[102,,]

Table 6. Toxicity Equivalency Factors for Chlorinated Dioxins, Furans and PAHs.

Compound	TEF
2,3,7,8-TCDD	1.0
2,3,7,8-PeCDD	0.5
2,3,7,8-HxCDD	0.1
2,3,7,8-HpCDD	0.01
OCDD	0.001
Other CDDs	0

Table 7. Toxicity Equivalency Factors for Chlorinated Furans

Compound	TEF
2,3,7,8-TCDF	0.1
1,2,3,7,8-PeCDF	0.5
2,3,4,7,8-PeCDF	0.05
2,3,7,8-HxCDF	0.1
2,3,7,8-HpCDF	0.01
OCDF	0.001
Other CDFs	0

Table 8. Toxicity Equivalency Factors for Carcinogenic Polycyclic Aromatic Hydrocarbons

Compound	TEF
Benzo(a)pyrene	1.0
Benz(a)anthracene	0.1
Benzo(b)fluoranthene	0.1
Benzo(k)fluoranthene	0.01
Chrysene	0.001
Dibenz(a,h)anthracene	1.0
Indeno(1,2,3-c,d)pyrene	0.1

Pentachlorophenol:

Pentachlorophenol (PCP) is a synthetic organic biocide (Figure 11), made from other chemicals, and does not occur naturally in the environment.[3] Since 1984, the EPA has restricted the purchase and use of pentachlorophenol to certified applicators[3] and regulatory limits (Table 9). Pentachlorophenol can exist as technical grade pentachlorophenol or as the sodium salt of pentachlorophenol. The sodium salt is hydrophilic but pentachlorophenol is hydrophobic. The two forms have different physical properties but have similar toxic effects.[3] Humans are generally exposed to technical-grade pentachlorophenol, which usually contains such toxic impurities as dioxins.[103,104,105,106,107] Pentachlorophenol was widely used as a wood preservative. Today it is used industrially as a wood preservative for power line poles, cross arms, fence posts, and railroad ties.[108]

The wastewater treatment sludge generated from wood preserving processes that use creosote and/or pentachlorophenol is listed by EPA as a hazardous waste. EPA defines these as K001 sludges.[109] Normally microorganisms break down many of the chemicals which are readily biodegradable, however certain higher molecular weight compounds such as PCP, resist biological breakdown and persist in the contaminated environments.[110] Pentachlorophenol and creosote are mutagenic in soil sludge even one year after application.[111]

Humans can be exposed to very low levels of pentachlorophenol through indoor and outdoor air, food, soil, and drinking water. Exposure may also result from dermal contact

with wood treated with preservatives that contain pentachlorophenol.[112] Pentachlorophenol is readily absorbed following oral or inhalation exposure and is widely and rapidly distributed throughout the body.[113] Absorption efficiency for inhalation exposure appears to be in the range of 70 to 88%.[114] PCP bio-accumulates mainly in the human adipose tissue.[115] PCP has a calculated half-life of 16 days.[116] An animal (broiler chickens) study conducted one year later resulted in accumulation of PCP in the kidney, liver, heart, leg, breast, gizzard, and fat (Stedman 1980). The major storage sites for PCP in humans are renal and hepatic tissues. [143]

Pentachlorophenol is extremely toxic when ingested by humans; the probable oral lethal dose is 50 to 500 mg/kg (1 teaspoon to 1 ounce) for a 70-kg person.[117] The scientific literature is full of examples with case reports describing PCP poisonings.[118,119,120,121,122,123,124,125,126,127] In fact as early as 1969, Robson reported that of the 51 cases on PCP poisonings, 30 resulted in death. Documented symptoms to acute exposures to toxic levels of pentachlorophenol consist of effects to the skin[128] eyes, mouth, liver, kidneys, blood, lungs, nervous system, immune system, pancreas, and gastrointestinal tracts.[118,119,120,121,122,123,124,125,126,127] Acute exposure also leads to increased basal metabolic rate with a subsequent increase in body temperature, respiratory rate, and heart rate.[129] Extreme exposure leads to death, which is preceded, by progressive neuromuscular weakness, convulsions, and cardiac arrest.[129]

Long-term exposure to low levels of PCP in humans can cause damage to the skin[130,131] liver,[132] kidneys,[133] cardiovascular,[134] immune,[116,132,135,136,137,138] and nervous system.[101]

Chronic exposure to PCP is associated with abnormalities, which may persist years after exposure.[131]

Studies in animals also suggest that the endocrine system,[139] reproductive,[140] and immune system[135] can also be damaged following long-term exposure to low levels of pentachlorophenol. All of these effects show a positive dose-response.

In 1977, a study examining 18 workers with PCP exposure had a mean PCP level of 5.1 ppm.[133] The study suggested that PCP exposure reduced both glomerular filtration rate and tubular function. The study suggested that the workers must have had "heavy chronic exposures to PCP."

In 1977, an industrial health survey of employees in a wood preservative plant in which coal tar creosote, coal tar, and pentachlorophenol were the main treatments used, cardiovascular effects, including increased diastolic blood pressure, were noted in 21% (24/113) of the employees examined.[134]

Dermal lesions have been reported for humans chronically exposed to pentachlorophenol-treated wood.[113,130,131] In one study of occupationally-exposed men, skin rash was recorded for 21.6% of the exposed and 28.3% of the unexposed. However, on physical examination 20.5% of the exposed presented with skin rash while only 16.4% of the unexposed had skin rash. This suggests that skin rash in the exposed is of longer duration

and perhaps more severe although no information on severity was reported compared to the same disorder in unexposed persons.[141]

In 1980, a study by Klemmer, reported a mean serum level of PCP in wood treatment workers to be 3.78 for open vat treaters and 1.72 for pressure tank treaters. PCP was associated with increased number of banded leukocytes, basophils, increased blood plasma cholinesterase, increased alkaline phosphatase and decreased serum calcium after controlling for the effects of age and ethnicity. Despite increases in clinical diagnosis of infections of the skin, dermatitis, chronic sinusitis, conjunctivitis, and hyperuricemia; the authors dismiss all health effects even though PCP levels were greatly increased.[142] A similar occupational study of wood-treating workers in Hawaii showed increased serum protein levels, higher systolic blood pressure and heart rate compared to controls.[141] These reports are from industry-funded sources where the incentive is clearly to document the safety of the material under study. Yet these studies indicate the potential for serious health effects.

PCP is toxic to our immune system.[143] Lang, 1981 reported that their most striking finding was that both analytic grade and technical grade PCP preparations caused immunosuppressive effects, indicating that PCP is immununotoxic to human immunicompetent cells by itself.[137] McConnachie also reported that individuals who were exposed to PCP treated log homes revealed activated T cells, autoimmunity, functional immunsuppression, and B cell dysregulation. In addition, the author reported all exposed subjects experienced an excessive incidence and persistence of cold and flu